cept not only to anterior but also posterior mitral (Figure 1, *B*) and aortic (Figure 1, *C* and *D*) periprosthetic leaks.[2] During the last decade, we have maintained that these alternative methods of repair with healthy autologous tissues are superior to direct suture closure of the leak. It is gratifying to see that others have started to use this technique with good results.

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Reply to the Editor:

We appreciate Dr Konstantinov for bringing his report to our attention. A MEDLINE search on the keywords prosthetic valve and paravalvular leak yielded 119 articles since 1966 but did not include his reference from the *Journal of Heart Valve Disease.* We apologize for overlooking it.

In heart surgery, as in life, there is indeed nothing new under the sun.

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Acute decrease of left ventricular mechanical dyssynchrony and improvement of contractile state and energy efficiency after left ventricular restoration

To the Editor:

We congratulate Dr Schreuder and colleagues[1] on their study of intraoperative left ventricular (LV) performance after endoventricular patch aneurysmectomy. In this series of 9 consecutive patients, the study demonstrates significant reduction in end-diastolic volume and increase in ejection fraction and LV energy efficiency.

However, we would counsel caution in this interpretation due to what we believe are three inherent flaws in the study methods.

First, the hemodynamic data highlight a significant difference in the heart rate before and after aneurysmectomy. It is widely established that an increase in heart rate proportionately alters LV systolic function and ejection fraction by virtue of the force-frequency relationship.[2]

Second, 7 of 9 patients underwent coronary artery bypass grafting, which may contribute to the increase in LV performance by reversal of hibernation. The authors discuss the potential independent effects of bypass grafting, but not the critical issue of the contribution of hibernating myocardium to LV function. We suspect that this factor may be responsible for augmented LV systolic function and ejection fraction.[3]

Third, there is no information regarding the severity of mitral regurgitation. This can cause spurious recording of increased LV performance due to inappropriate offloading of the left ventricle.[4]

Notwithstanding these issues, we believe that this is a valuable study contributing required evidence to support objective improvement in LV function with reverse remodelling surgery.

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Reply to the Editor:

We thank Dr Balacumaraswami and colleagues for their interest in our study on the acute effects of left ventricular (LV) restoration on cardiac performance. We prefer to answer in reverse order.

The reason for not giving any information on mitral regurgitation (MR) was the absence of significant perioperative MR in our patient group, all of whom underwent routine echocardiographic examinations. Concerning MR, McCarthy[1] referred to Di Donato and colleagues,[2] revealing that 10% of their patients had preoperative MR and 38% of their patients had had MR develop by 1 year after LV restoration, whereas immediately after surgery the patients were free from MR. When present, MR may offload the left ventricle; however, offloading will not change contractile state. Recently we[3] demonstrated that acute decrease in LV afterload by intra-aortic balloon pumping resulted in instantaneous increases in stroke volume but not in an increase of contractile state.

The second comment suggested that a significant improvement of LV systolic function and ejection fraction may have been attributable to recovery of hibernating myocardium in patients with additional coronary artery bypass grafting. Bax and colleagues[4] demonstrated in patients with ischemic cardiomyopathy undergoing CABG a reduction in wall motion abnormalities, based on hibernation, in 30% of the segments 3 months after bypass grafting. This suggests that in our acute LV restoration study, recovery from hibernation might have occurred; however, its contribution to an increase in contractile state could be doubted. Moreover, LV contractile state is commonly impaired or unchanged immediately after cardiopulmonary bypass.[5,6] In Table 3 of the LV restoration manuscript, we showed that LV mechanical dyssynchrony decreased significantly in the apical and midventricular segments, the areas of the LV restoration, whereas the dyssynchrony of the basal segments was unchanged.[7] The major finding of our study, the marked relationship between LV mechanical dyssynchrony and contractile state of the heart, however, is

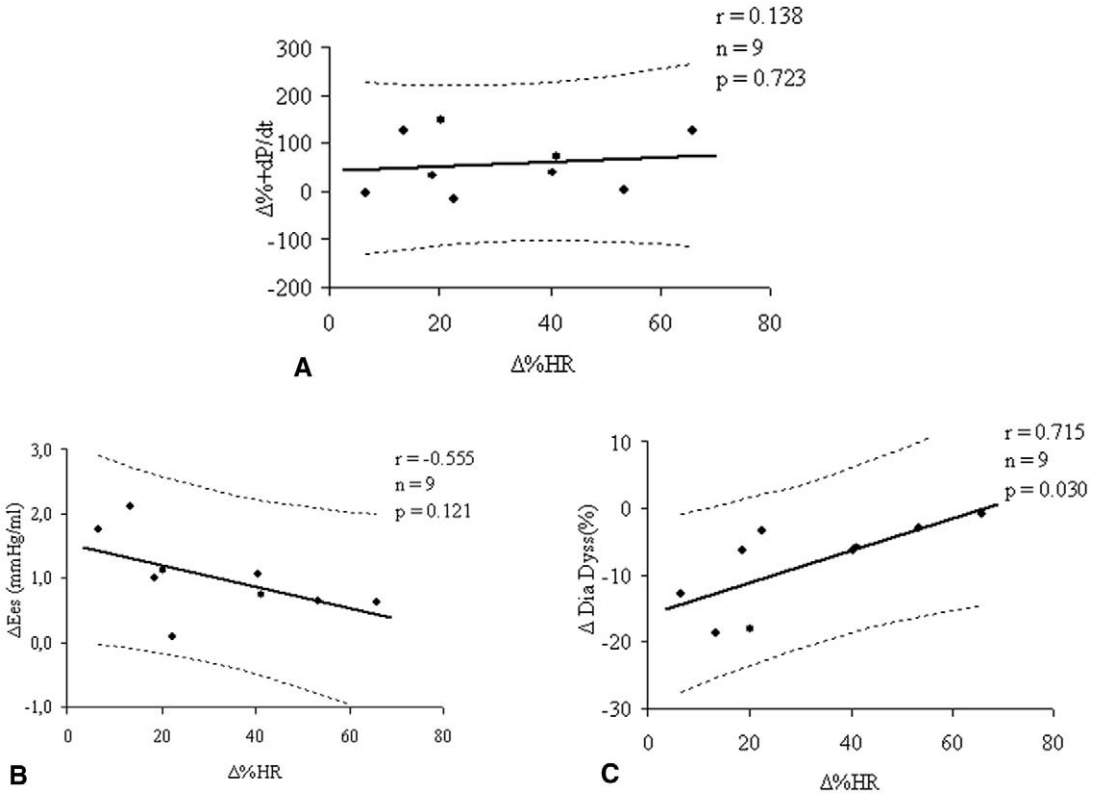

Figure 1. Regression diagrams showing in A, percent change in $+dP/dt_{max}$ versus percent change in heart rate *(Δ%HR);* in B, change in end-systolic elastance *(ΔEes)* versus Δ%HR; and in C, diastolic dyssynchrony change *(ΔDiaDyss)* versus Δ%HR. *Dotted lines* represent 95% prediction limits.

not affected by a possible decrease in mechanical dyssynchrony due to recovery from hibernation.

The first suggestion by Dr Balacumaraswami and colleagues concerned the significant increase in heart rate, present immediately after bypass, which might have increased contractile state on basis of the force-frequency relationship. Feldman and associates[8] showed that a heart rate increase of 60 beats/min increased $+dP/dt_{max}$ by 30% in healthy man, whereas an increase of 30 beats/min did not increase $+dP/dt_{max}$. In patients with dilated cardiomyopathy, however, a mean increase in heart rate of 55 beats/min from a baseline value of 82 beats/min did not result in any significant change in $+dP/dt_{max}$.

In our study, mean heart rate changed from 76 to 99 beats/min after left ventricular restoration in these patients with dilated hearts, whereas mean $+dP/dt_{max}$ increased by 41%.[7] Figure 1, *A,* shows the nonsignificant correlated force-frequency relationship from our study (r = 0.138, n = 9, P = .723). Figure 1, *B,* shows the relationship between change in heart rate and change in end-systolic elastance (Ees) due to LV restoration (r = −0.55, n = 9, P = .12). This suggests that heart rate merely increased as a compensatory mechanism in patients, characterized by a small increase in contractile state (Ees) and small decrease in systolic dyssynchrony, to maintain cardiac index. We[9] previously indicated that stroke volume could only be maintained by decreases in LV mechanical dyssynchrony in patients undergoing partial left ventriculectomy. This reasoning is confirmed by the significant correlation between heart rate change and dyssynchrony change (Figure 1, *C,* r = 0.715, P = .03) in the patients undergoing the LV restoration. In case mechanical resynchronization was suboptimal and end-diastolic volume decrease was significant, a heart rate increase may have compensated for decreased stroke volume (cardiac index). In the LV restoration manuscript, we indicated that heart rate increase correlated significantly with end-diastolic volume decrease (r = −0.679, P = .044) and that contractile state (Ees) was markedly inversely related (P <.00001) to LV mechanical dyssynchrony.

In conclusion, frequency did not correlate with force ($+dP/dt_{max}$) in our patient group undergoing LV restoration, but was significantly related to decrease of LV volume and LV mechanical dyssynchrony, emphasizing the importance of aneurysmectomy combined with geometric remodeling to achieve maximal ventricular mechanical synchrony.

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